ABC of oxygen

Diving and oxygen

Peter Wilmshurst

All organisms require oxygen for metabolism, but the oxygen in water is unavailable to mammals. Divers (and diving mammals such as whales and seals) are entirely dependent on the oxygen carried in the air in their lungs or their gas supply. Divers also have a paradoxical problem with oxygen. At higher partial pressures oxygen causes acute toxicity leading to convulsions. To understand the diver's narrow knife edge between fatal hypoxia and fatal hyperoxia we need to recall some of the physical properties of gases.

Physics

At sea level atmospheric pressure is 1 bar absolute (1 standard atmosphere = 101 kPa = 1.013 bars). The weight of the atmosphere exerts a pressure which will support a column of water 10 m high; 10 m under water the pressure on a diver is 200 kPa. The volume of gas in an early diving bell full of air at sea level is halved at 10 m according to Boyle's law; at 20 m pressure is 300 kPa absolute and the gas is compressed into one third the volume.

Dry air is composed of roughly 21% oxygen, 78% nitrogen, and 1% other gases. According to Dalton's law the partial pressure of oxygen at any depth will be 21% of the total pressure exerted by the air and the partial pressure of nitrogen will be 78% of total pressure.

Gases dissolve in the liquid with which they are in contact. Nitrogen is fat soluble and at sea level we have several litres dissolved in our bodies. If the partial pressure of nitrogen is doubled (by breathing air at 10 m depth) for long enough for equilibration to take place we will contain twice as many dissolved nitrogen molecules as at sea level.

The effect of the increased partial pressures of oxygen is more complex. Doubling our inspired partial pressure of oxygen doubles the amount of oxygen in solution but does not double the amount of oxygen in the body since a large part of our oxygen content is bound to oxygen carrying pigments. The haemoglobin in arterial blood is virtually saturated at an inspired partial pressure of oxygen (F_{IO_2}) of 21 kPa, and increasing the partial pressure of oxygen has little effect on the amount of oxygen bound to haemoglobin.

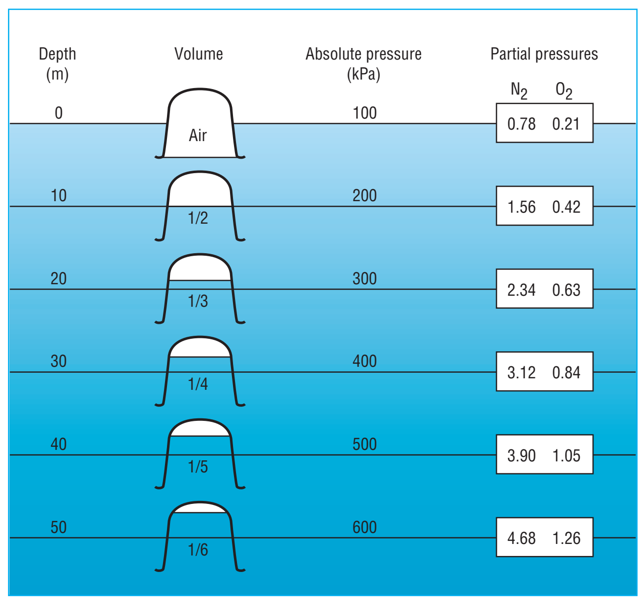
Breath hold diving

An average healthy person with no special training can hold his (or her) breath for about half a minute. During the breath hold the oxygen content of tissues decreases, but the breath hold is broken as a result of carbon dioxide production and resulting acidosis, which stimulates the respiratory centre. With practice you can resist the stimulus to breathe for longer but it remains carbon dioxide accumulation that causes release of the breath hold.

The breath hold can be extended further by hyperventilation immediately beforehand. Hyperventilation has little effect on the oxygen content of the body but blows off carbon dioxide so that you start with a higher cerebrospinal fluid pH. Hyperventilation does not alter the rates of oxygen consumption and carbon dioxide production, but the lower initial carbon dioxide content means that the hypoxic stimulus

A dive to 30 m for 20 minutes puts the scuba diver at risk of nitrogen narcosis and decompression illness. The elephant seal can dive to 1 km for 1 hour without risk of either condition

The pressure on a diver increases by 100 kPa for every 10 m he or she descends

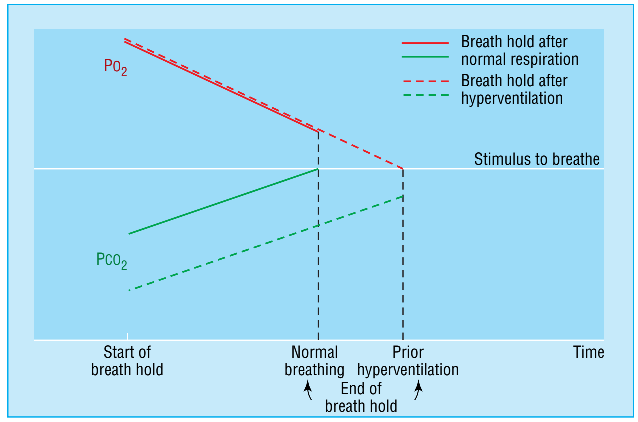


Effect of depth on partial pressures of nitrogen and oxygen

Ama divers do repeated breath hold dives with little time in between for recovery triggers respiration before the pH of the cerebrospinal fluid falls enough to do so. It may be possible to hold a breath for over 5 minutes by hyperventilation on 100% oxygen. The hyperventilation reduces the body's carbon dioxide content but does not affect oxygen content much, but the F_{IO_2} of 100 kPa considerably increases the total oxygen content.

Hyperventilation before diving enables breath hold divers to stay down longer but is very dangerous. The diver starts with a low carbon dioxide content, a high pH, and a normal oxygen tension. During descent to, say, 30 m, the pressure increases fourfold, compressing the airspaces to one quarter their surface volume (from total lung capacity of 6 l to 1.5 l, near residual volume). The partial pressures of oxygen and nitrogen in the alveoli also increase fourfold and produce corresponding increases in arterial and tissue gas tensions. The alveolar carbon dioxide pressure does not change much because there is little carbon dioxide in the lungs at this point and the body has considerable buffering capacity. During the dive oxygen is consumed and carbon dioxide is produced. Because of the hyperventilation the diver does not feel the need to breathe until the arterial oxygen tension has fallen to levels which stimulate the carotid chemoreceptors. As the diver ascends hydrostatic pressure is reduced fourfold with a fourfold reduction in oxygen tensions in alveolar gas, arterial blood, and tissues. The rapidly falling cerebral oxygen pressure may be inadequate for consciousness to be maintained and the diver could drown during ascent.

The danger of hyperventilation applies to all breath hold divers, including snorkel divers and people swimming lengths underwater in pools. The reduction in oxygen pressure when coming to the surface from the bottom of a 2 m deep pool can be enough to cause unconsciousness, and some children have died this way.



Effect of hyperventilation on breath holding

Bubbles formed on decompression are visible in tear fluid beneath contact lens

Amateur scuba divers

Scuba and surface supplied diving

Air

The most available and cheapest gas to use for scuba (self contained underwater breathing apparatus) or surface supplied diving is air. It can be compressed easily using simple machines. Air is less likely to produce a fatal mixture than gas mixing but has several disadvantages. With the high partial pressures at depth nitrogen affects the function of cell membranes causing nitrogen narcosis. Mild impairment of intellectual function may occur at only 30 m, with progressive impairment of function as the diver descends and unconsciousness at depths near 100 m.

The nitrogen that dissolves in the tissues at depth also needs to be liberated on ascent or decompression. Because nitrogen is highly soluble a large volume of gas may be involved. If the rate of decompression (ascent) is too rapid large amounts of bubbles are liberated from the supersaturated tissues. For most air dives the rate of ascent should be no faster than 10-15 m/min. For some deep or long dives decompression stops are performed to allow gas to be released without excessive formation of bubbles in vulnerable tissues. Small amounts of bubbles are common after innocuous dives, but too many bubbles or bubbles in the wrong place cause decompression illness. Even breath hold divers who repeatedly dive to 20-30 m for a couple of minutes with shorter surface breaks between can accumulate enough nitrogen to develop decompression illness at the end of the day.

Nitrogen is a relatively dense gas, which makes the work of breathing at 30 m depth twice as great as at the surface. A breathing system using air requires that the exhaust gas (low in oxygen and high in carbon dioxide and nitrogen) be liberated

Characteristics of types of decompression illness

Causes	Neurological	Cardiorespiratory	Skin	Joint
Paradoxical gas embolism—cardiothoracic shunt	Severe	Mild	Severe	None
Arterial gas embolism after pulmonary barotrauma:				
Lung disease	Mild	Mild	None	None
Rapid ascent	Mild	Mild	None	None
Gas nucleation caused by unsafe decompression profile	Mild	Mild	None	Severe

as bubbles. This can be a problem in military covert operations or defusing naval mines with acoustic sensors.

Oxygen

Several approaches have been developed to deal with the problems of nitrogen. The first was to breathe 100% oxygen using a rebreathing system with a carbon dioxide absorber. The diver breathes into and out of a bellows-like counterlung with the oxygen supply topped up from a cylinder and absorption of carbon dioxide.

Divers breathing pure oxygen need to carry much smaller amounts of gas and produce no bubbles, but there are problems, some of which can be fatal. When a diver starts breathing from an oxygen rebreather the fraction of inspired nitrogen is zero. The diver's body contains several litres of dissolved nitrogen, and the pressure gradient causes this nitrogen to pass back to the lung and into the counterlung. The oxygen is consumed, carbon dioxide is removed, and nitrogen accumulates, gradually reducing the percentage of oxygen in the counterlung. This can lead to unconsciousness. Flushing the system with pure oxygen periodically overcomes this problem, but high partial pressures of oxygen increase blood pressure and reduce heart rate. These effects are, however, small and reversible.

Prolonged breathing of a gas with an FiO_2 greater than 60 kPa can lead to pulmonary toxicity and eventually irreversible pulmonary fibrosis, but this takes many hours or days. At an FiO_2 greater than 160 kPa acute oxygen toxicity can occur within minutes causing convulsions with little or no warning. A convulsion underwater is usually fatal. The higher the FiO_2 the greater the risk. Breathing air containing 21% oxygen risks acute oxygen toxicity at depths greater than 66 m; breathing 100% oxygen there is a risk of convulsion at only 6 m.

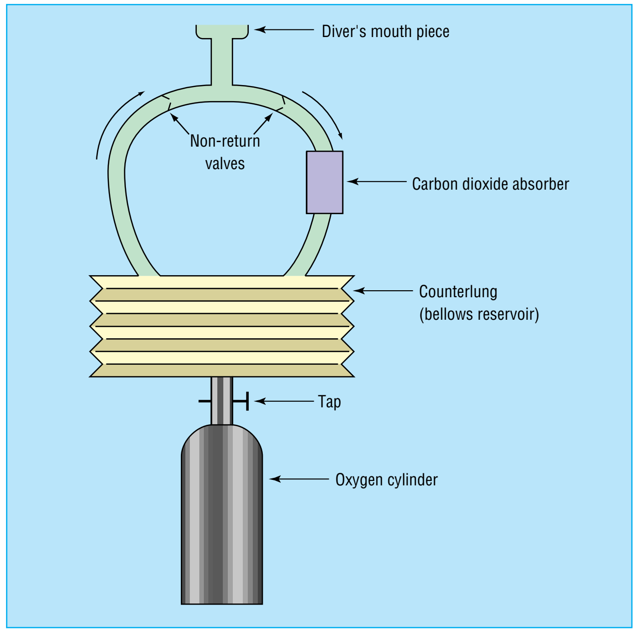
Nitrox

Amateur divers increasingly breathe a nitrogen-oxygen (nitrox) mixture. Almost any mixture can be made, but a typical example is nitrox 40, which consists of 40% oxygen and 60% nitrogen. (The number always denotes the percentage of oxygen.) The reduced nitrogen content compared with air increases the time the diver can stay on the bottom without getting decompression illness on surfacing. The trade off is that there is a risk of convulsion from acute oxygen toxicity if the diver descends too deep; for nitrox 40 that would be deeper than 30 m.

Mixed gases

For dives deeper than 66 m the gas mixture should contain less than 21% oxygen to avoid the risk of acute oxygen toxicity. The general rule is to try to achieve a gas mixture giving an FiO_2 of about 140 kPa. At 130 m depth in the northern sector of the North Sea oil field, the ambient pressure is 1400 kPa, so the breathing mixture used contains 10% oxygen. On the deepest working dives, at depths greater than 600 m, ambient pressure is greater than 6100 kPa and the divers breathe gas mixtures containing about 2% oxygen to avoid acute oxygen toxicity. A lung full of gas containing 2% oxygen at 600 m contains about six times as many molecules of oxygen as a lung full of air at sea level. On deep dives the composition of the gas breathed is changed several times during descent and ascent.

Which gases should be used to dilute the oxygen on deep dives? The choice requires a compromise which takes into account the various properties of possible gases. Helium is commonly used with oxygen (heliox), even though helium is expensive and has a high thermal conductivity, which potentiates heat loss and can make hypothermia a serious

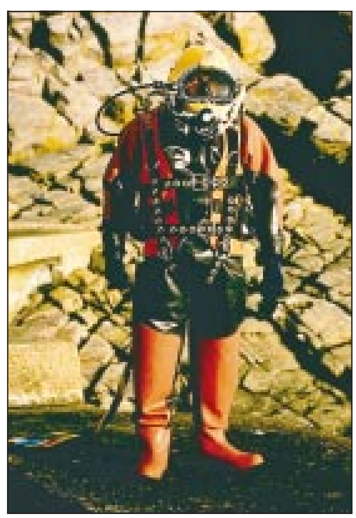


Oxygen rebreathers allow divers to breathe 100% oxygen but carbon dioxide accumulation can be a problem

Divers should always attempt to keep their FiO_2 below 160 kPa

Recompression facilities

An emergency 24 hour telephone number for diving emergencies exists at HM Dockyard, Portsmouth. It will advise on the nearest available recompression facility. Tel: 01705 818888.



Professional surface diver with umbilical gas supply, voice communication to helmet, and heating to suit possibility on deep dives. Helium molecules are small so that the work of breathing is low even at great depths. It is relatively insoluble in lipids, minimising bubble liberation on decompression. Its insolubility means that it lacks narcotic effects, but this unmasks another problem of diving deep, the high pressure nervous syndrome. This syndrome is believed to be the direct effect of pressure exciting neurones. Adding a small amount of a narcotic gas such as nitrogen can ameliorate some of the symptoms but this is not the entire answer and other experimental gases are used.

Symptoms of high pressure nervous syndrome

- Impaired intellectual function
- Tremor
- Myoclonus
- Fits

Amateur sport diving

Non-specialist doctors are unlikely to have much involvement with commercial divers, but most general practitioners will have amateur divers among their patients. Each year in Britain there are about 12 deaths and 100 cases of serious decompression illness requiring recompression. Most occur because divers failed to follow accepted safety precautions, equipment failed, or disease placed the diver at risk. Several organisations train sport divers in clubs and commercial schools. Instructors take new divers through basic theory and pool training to progressively more challenging and deeper open water dives. The trainee should be certified as competent before being allowed to undertake dives in the company of another diver without an instructor. Further training is needed before the qualified diver can progress to more adventurous diving.

Before anyone is allowed to start diving, and periodically when diving, they have to pass a diving medical examination to ensure freedom from diseases which might predispose to incapacity in the water or to diving related illnesses. The requirements for amateurs in all diving clubs in the United Kingdom are laid out in a common medical form.

Lung disease in divers is a particular problem. Significant lung disease which impairs exercise performance and the ability to cope with physically demanding conditions is obviously a contraindication to diving. Asymptomatic lung disease which does not affect exercise capacity is also a problem. Any lung disease which causes generalised or localised gas trapping (such as emphysema, bullae, cavities) may predispose to pulmonary barotrauma during ascent, even when the ascent rate is less than 10-15 m/min. During ascent from a dive the gas in a bulla increases as ambient pressure is reduced. If the bulla cannot empty adequately during the ascent it will burst causing local lung damage, pneumothorax, surgical emphysema, or arterial gas embolism. Gas in a pneumothorax will expand as pressure is reduced, causing a tension pneumothorax.

In the United Kingdom people with mild asthma who satisfy criteria laid down by the United Kingdom Sport Diving Medical Committee may be approved to dive by a medical referee. In some countries anyone with a history of asthma, even childhood asthma decades before, is not permitted to dive. Ironically those countries allow smokers to dive, yet a long term heavy smoker with evidence of small airways disease on flow-volume loops is probably at greater risk of pulmonary barotrauma than a patient with mild asthma who has never smoked.

There are also medical standards for non-respiratory diseases. People are advised not to dive if they have a condition which may cause incapacity in the water—for example, epilepsy or cardiac arrythmias—or predispose to diving related diseases. Intracardiac shunts predispose to decompression illness and hypertension predisposes to diving induced pulmonary oedema.

The picture of an ama diver was provided by Rex Features.

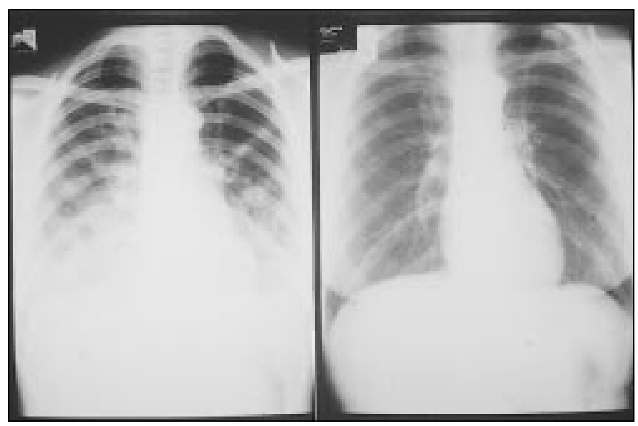
Courses on diving and hyperbaric medicine

Institute of Naval Medicine, Alverstoke, Gosport, Hampshire PO12 2DL (tel: 01705 768091)
Hyperbaric Medicine Unit, Aberdeen Royal Infirmary, Aberdeen AB25 2ZN (tel: 01224 681818)

British amateur scuba diving organisations

British Sub-Aqua Club, Telford's Quay, Ellesmere Port, Cheshire L65 4FY (tel: 0151 350 6200)
Scottish Sub-Aqua Club, Cockburn Centre, 40 Bogmoor Place, Glasgow G51 4TQ (tel: 0141 425 1021)
Sub-Aqua Association, Bear Brand Complex, Allerton Road, Liverpool L25 7SF (tel: 0151 428 9888)

These organisations have a panel of doctors throughout the United Kingdom (and a few places elsewhere) who are diving medical referees and who will advise on fitness to dive and about diving related diseases



Diving induced pulmonary oedema (left) which resolved with no treatment after diver was removed from water (right)

Further reading

- *Sport diving. The British Sub-Aqua Club diving manual.* 11th ed. London: Stanley Paul, 1993.
- Bove AA, Davis JC. *Diving medicine.* 2nd ed. Philadelphia: WB Saunders, 1990.

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